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Autor*innen/Author(s): Martin Diewald, Tina Baier, Wiebke Schulz and Reinhard Schunck

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Zusätzliche Informationen/Additional information:
The author can be contacted at: wschulz@uni-bremen.de

Status Attainment and Social Mobility
How can Genetics Contribute to an Understanding of their Causes?

Martin Diewald, Tina Baier, Wiebke Schulz and Reinhard Schunck

Abstract

This paper discusses why and how the consideration of inter-individual genetic variation can enhance the explanatory power of sociological inquiries of status attainment and social stratification. We argue that accounting for genetic variation may help to address longstanding and in some cases overlooked causality problems in explaining the emergence of social inequalities – problems which may interfere with both implicit and explicit interpretations of a society as "open" or "closed," as meritocratic or non-meritocratic. We discuss the basic methodological tenets of genetically informative research (Sect. 2) and provide empirical examples and theoretical conceptualizations on how genetic variation contributes to status attainment (Sect. 3). This is followed by a discussion of gene-environment interplay in relation to more abstract ideas about social mechanisms that generate inequality, touching on normative implications of these ideas as well as considerations from a social justice perspective (Sect. 4). Finally, we briefly review the potential benefits as well as pitfalls of incorporating genetic influences into sociological explanations of status attainment. As we will argue, understanding how social influences impinge on the individual and how genes influence our lives requires sophisticated research designs based on sound sociological theory and methodology (Sect. 5). **(← p. 371)**

Keywords: Social Stratification, Social Mobility, Inequality, Genetics, Behavioral Genetics, Heritability, Twin Study, Extended Twin Family Design

Status Attainment und Soziale Mobilität
Wie kann Genetik zu einem Verständnis ihrer Ursachen beitragen?

Zusammenfassung

Dieser Beitrag legt dar, wie die Berücksichtigung genetischer Variation die Erklärungskraft soziologischer Untersuchungen zu Status Attainment und sozialer Ungleichheit verbessern kann. Die Berücksichtigung genetischer Variation kann helfen, Probleme kausaler Schlüsse bei der Erklärung sozialer Ungleichheit zu mindern, die für eine implizite oder explizite Interpretation einer Gesellschaft als „offen“ oder „geschlossen“, als meritokratisch oder nicht meritokratisch ausschlaggebend sein können. Nach der Einleitung stellen wir die methodologischen Grundlagen verhaltensgenetischer und genetisch informativer Forschung dar (Abschn. 2) und zeigen theoretische Mechanismen und empirische Beispiele auf, wie genetische Variation Status Attainment beeinflussen kann (Abschn. 3). Anschließend werden die Grundlagen von Gen-Umwelt-Interaktionen diskutiert, insbesondere im Hinblick auf theoretische Überlegungen zur Genese und Bewertung sozialer Ungleichheit (Abschn. 4). Im letzten Teil stellen wir mögliche Vorteile und Fallstricke der Einbeziehung genetischer Variation in soziologische Erklärungen zu Status Attainment und sozialer Ungleichheit dar. Um zu verstehen, wie soziale und genetische Faktoren miteinander wirken und das Leben beeinflussen, braucht es anspruchsvolle Forschungsdesigns auf der Grundlage solider soziologischer Theorie und Methodologie (Abschn. 5).

Schlüsselwörter: Stratifizierung, Soziale Mobilität, Ungleichheit, Genetik, Verhaltensgenetik, Erblichkeit, Zwillingstudie, Extended Twin Family Design

1 Introduction

A central goal of sociological research is to explore how society shapes the individual life course and structures individual opportunities. But how should "the individual" exposed to societal influences be conceptualized? Social stratification and inequality research has addressed this question by focusing on social origins, which are generally defined in terms of parental social class, status, resources, and family structure. In this contribution, we demonstrate why and how the consideration of inter-individual genetic variation over and above social origin can enhance the explanatory power of sociological and particularly social mobility research. Furthermore, we discuss how this endeavor might help to address longstanding and in some cases overlooked causality problems in explaining the emergence of social inequalities—problems which may interfere with both implicit and explicit interpretations of a society as "open" or "closed," as meritocratic or non-meritocratic. Moreover, this discussion provides an example of how genetically informative research can contribute more generally to established sociological theories and research. **(← p. 372)**

Sociology has developed several approaches to investigate the relationship between social origin and destination and the pathways that mediate between them. Social mobility research tends to proceed by studying associations between social origins and social outcomes, be they in social class (Erikson and Goldthorpe 1992), socioeconomic status, or material resources. The basic status attainment model developed by Blau and Duncan (1967) enlarged the connection between social origin and destination to include two additional pathways: One between social origin and education and another between education and destination. The idea of this model was to test whether status attainment based on social origins was being replaced by meritocracies based on education, which channel social mobility through educational and vocational degrees. The weaker the direct path from origin to destination and from origin to education and the greater the influence of education on destination, the more open in terms of the equality of opportunity provided to its citizens is a society assumed to be (Breen and Jonsson 2005). However, this interpretation is often dismissed as invalid since a strong family influence may also entail meritocratic processes, e.g. skill formation or motivation (Saunders 2002).

Over the years, this basic status attainment model has been extended in a number of ways, foremost through the Wisconsin model, which integrated interpersonal influences and aspirations as mediating mechanisms and later cognitive and non-cognitive skills (i.e., Haller and Portes 1973; Hauser et al. 2000; Heckman 2006). Life course research has added ever

more detailed pathways from social origin to destination in different phases of life that are affected by a wide range of life experiences and social contexts, which are beyond the scope of this article to discuss in detail.

Nevertheless, even with relatively comprehensive measurements of social origins and skills, the overall impact of social origins and individual characteristics on educational and status attainment is still not fully understood, and the relative contributions of both may be biased by unmeasured characteristics (Jencks and Tach 2006; Smeeding et al. 2011). Educational certificates are not simply an indicator of achievement and meritocratic selection, but may reflect social closure as well (Collins 1979). Conversely, residual impacts of the family of origin in status attainment models, not to speak of social mobility tables, may reflect not only ascription but also ability and effort. In other words, the research on individual characteristics and social influences that link social origin and destination is still ongoing and far from complete.

So far most of the studies on this subject have focused on unequal chances between members of different families. Status attainment models assume that children from the same family are influenced in the same ways and to the same degree by family processes and resources. Much less attention has been paid to possible inequalities created within families. Sibling research shows that the assumption of equality between siblings may need to be reconsidered, with attainment correlations between siblings of only about 0.5 (Hauser and Wong 1989; Downey 1995; Conley 2004). Thus, within-family differences in attainment may indeed constitute an important part of a society's inequality structure—yet one that has gone largely ignored so far in the research. The obvious differences between children from the same family point to the complex familial dynamics structuring unequal life opportunities far beyond those usually captured in status attainment research. **(← p. 373)**

Moreover, parents not only pass on resources and experiences to their children, but also their genetic predispositions. Because of this, inequalities exist between individuals from birth on, not only in their social origins but also in their genetic endowments, negating the assumption underlying much of the standard social research that human beings are a "blank slate" at birth. As some sociologists have already suggested, acknowledging the role of genetics and incorporating it into sociological research designs may help to overcome the incompleteness and ambiguity of model parameters as measures of achievement versus ascription (Nielsen 2006; Freese 2008; Adkins and Vaisey 2009).

In this paper, we try to develop the arguments underlying this suggestion a bit further. We start with a general discussion of what the heritability of social outcomes implies, including a brief introduction to the methodological tenets of genetically informative research, to address the question of how genetic variations shape social forces, and conversely, how social forces shape genetic influences (Sect. 2). Section 3 explores the implications of this discussion for status attainment research and life chances at large: The genetic dimension contributes to a more complete and useful definition of the family of origin than purely social conceptualizations and allows addressing the interplay between genes and social environment. In Sect. 4, we discuss processes of gene–environment interplay in relation to more abstract ideas about social mechanisms that generate inequality. This discussion also touches on normative implications of these ideas as well as considerations from a social justice perspective. Finally, we briefly review the potential benefits as well as pitfalls of incorporating genetic influences into sociological explanations of status attainment. As we will argue, understanding how social influences impinge on the individual and how genes influence our lives requires sophisticated research designs based on sound sociological theory and methodology (Sect. 5). As we will demonstrate, considering both social and genetic factors jointly in such a way is also valuable for demographic research and the explanation of fertility (Kohler et al. 1999; Kohler and Rodgers 2003) and mortality (Vaupel 2004; Carey and Vaupel 2005).

2 Genetic Influences and Social Science Research

2.1 Genetically Informative Research Designs: Methodological Tenets

There is increasing evidence that genetic variation plays an important role in explaining differences in individual outcomes (e.g. Turkheimer 2000; Freese 2008). However, integrating genetic influences empirically is a challenging endeavor. This section gives a broad overview of the methodologies and research designs rooted in the field of behavioral genetics that allow for a genetically sensitive investigation of social scientific research questions (for a more detailed discussion see Kim 2009; Plomin et al. 2013). There are two basic strategies for investigating how the interplay between nature and nurture influences life outcomes: quantitative and molecular genetic approaches. The first is to use behavioral genetic designs in which family members with different degrees of genetic and/or environmental similarity are compared, and the second is to include molecular genetic information. Quantitative **(← p.**

374) approaches can be used to quantify the extent to which variation in a phenotype—any observable trait or characteristic of an organism—is related to genetic variation as a whole without knowing about which specific genetic variants are at work. Molecular genetic approaches offer techniques that can be used to analyze how and to which degree specific genetic variants directly affect phenotypes, which might be a smaller or bigger part of the overall genetic influence. These two approaches are not mutually exclusive but rather complementary strategies (Kendler 2001; Weinstein et al. 2008). We will discuss both approaches, but with a stronger emphasis on quantitative genetic approaches as they seem currently better suited to provide a more comprehensive picture of genetic influences on mobility outcomes. Additionally, we point to the importance of the gene and environment interplay that needs to be considered within these two approaches.

2.1.1 Quantitative Genetics

Quantitative genetics offers a means of indirectly assessing the relative contributions of genetic and non-genetic (i.e., environmental) factors in observable phenotypic variation by looking at phenotypic similarity in relatives with known (and different) *average* degrees of genetic relatedness.[1] The underlying idea is straightforward (Plomin and Daniels 2011): If a certain characteristic is influenced by genetic factors, relatives who are genetically more similar will be more similar in the characteristics of interest. This approach is best illustrated with the classical twin design, the "workhorse" of behavioral genetics (Plomin and Kosslyn 2001, p. 1154).[2] Monozygotic twins are genetically identical; dizygotic twins, in contrast, share—like "normal" siblings—on average only half of their DNA. But both mono- and dizygotic twins grow up under same, shared familial conditions, so that influences of the shared environment can be assumed to be the same.

This simple ACE model assumes that the trait under study (P, the phenotype) is produced through *additive* influences of alleles (A, the genotype), shared environmental factors (C), and non-shared environmental factors (E). With this model, we can estimate how much phenotypic variance is due to genetic variance and how much is due to environmental variance—that is, we can estimate *heritability*.[3] Total phenotypical variance (σ^2_p) is therefore

[1] It is important to note that these designs rely on average known degrees of relatedness. For instance, dizygotic twins share 50% of their genes on average. A particular dizygotic twin pair may also share more, or fewer, genes.
[2] There are also other types of genetically informative designs (i.e. the adoption design). All of them follow the same idea and use information on known degrees of genetic and/or environmental similarity (for an overview see, i.e.; Plomin et al. (2013)).
[3] This is called narrow-sense heritability, because it only estimates the proportion of variance due to additive

assumed to being the sum of the variance components of A, C, and E.

$$\sigma^2_P = \sigma^2_A + \sigma^2_C + \sigma^2_E \quad (1)$$

(← p. 375)

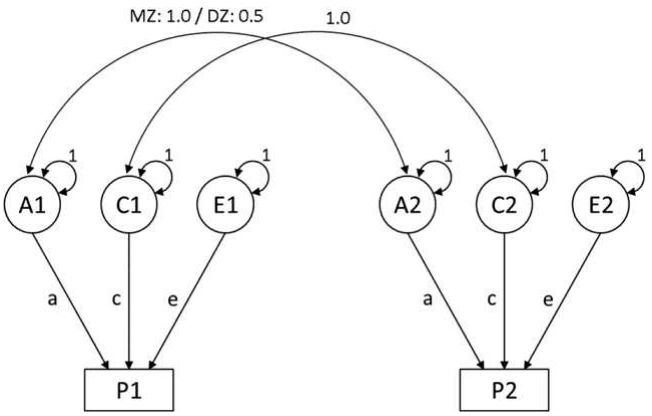

Figure 1. ACE path diagram, including expected correlations among MZ and DZ twins

These variance components can be estimated, for instance, via structural equation modeling, as displayed in Figure 1.

Heritability in the narrow sense (h^2) is defined as the share of the total variance attributable to additive variance of additive genetic effects[4]

$$h^2 = \frac{\sigma^2_A}{\sigma^2_A + \sigma^2_C + \sigma^2_E} \quad (2)$$

For instance, if studies estimate heritability in IQ to being around 50 to 60% (Bouchard and McGue 1981; Deary et al. 2009; Plomin et al. 2008), this means that of the total observable variance in IQ, this percentage is based on variance in additive genetic factors. The model is simplistic and relies on rather strict assumptions. It ignores nonadditive effects, i.e. that alleles can interact with each other (I, epistasis) or suppress other alleles (D, dominance deviations) or that genes and environment may correlate or interact (see below) (e.g. Plomin et al. 2013). Additionally, it is assumed that there is no assortative mating of parents, and that MZ and DZ

genetic effects (Purcell 2013, p. 381).

[4] Heritability can also be estimated through mixed effects (multilevel) models and DeFries-Fulker models.

twins grow up under similar conditions (the so-called "equal environment assumption") and are treated equally by their social environment (Derks et al. 2006; Scarr and Carter Saltzman 1979). If there are non-additive genetic effects or, more generally, if any of these assumptions are violated, estimates of heritability will be biased (Visscher et al. 2008). The main reason for imposing these strict assumptions lies in data limitations. More complex models require data on more than just twins.

Besides estimating the relative influence of genetic and environmental factors on individual traits, multivariate models can also be used to assess the extent to which variance in different phenotypes is due to the same genetic or environmental factors (Posthuma 2009; Purcell 2013, p. 393).

Taking the aforementioned assumptions and limitations into account, it becomes clear that only by properly accounting for social influences can genetic factors be estimated accurately—and vice versa. The "extended twin family design" (ETFD) (see, i.e.; Keller et al. 2009) is a promising research strategy as it includes not only **(← p. 376)** mono- and dizygotic twins but also various other types of family members. These differences in kinship can be exploited to provide more rigorous estimates of genetic influences (Posthuma and Boosmsa 2000; Coventry and Keller 2005). Adopting the ETFD makes it possible to relax assumptions and thereby capture the different influences more accurately. In particular, the ETFD can help to distinguish the effects of shared and non-shared environments and thus to pinpoint different causes of a given outcome.

Interpreting heritability Although estimating heritability has been a major focus of behavioral quantitative genetics in recent decades, this line of research is relatively new in other social sciences and may be misunderstood. Before we come to a substantial interpretation of heritability estimates (paragraph 3) we first discuss the underlying concept of heritability estimates and their limitations (Shanahan et al. 2003; Turkheimer 1998; Visscher et al. 2008; Plomin et al. 2013). First, it is important to note that heritability estimates are population- and time-specific (Plomin et al. 2013, p. 92). A high heritability estimate of approximately 80% in height (Carmichael and McGue 1995), for instance, does not indicate that the environment is unimportant. Height has increased substantially in Western societies over the twentieth century due to environmental factors including nutrition (Shanahan et al. 2003, p. 608). Heritability estimates refer to a specific social system, point in time and population (or sample). They can therefore be "expected to vary across societies, historical periods and social contexts" (Nielsen 2006, p. 208). The fact that heritability estimates vary

according to environmental influences is an important clue towards the interplay of environment and genes. The consistently higher heritability in educational achievements for men as compared to women is one finding that suggests that context influences work differently for the realization of the genetic dispositions of men and women (Branigan et al. 2013). Second, high heritability therefore does not imply that environmental factors cannot mitigate or even override genetic effects, as the height example indicates. Third, heritability estimates cannot be treated as "fixed" properties of a given trait: Any increase in environmental differences in a sample automatically decreases the extent to which genetic factors contribute to the variation, and vice versa, as Eq. (2) indicates. Fourth, heritability estimates are population parameters, and cannot be used to explain genotype–phenotype links at the individual level (Shanahan et al. 2003, p. 607). A heritability estimate of 0.8 for height means that on average 80% of observed differences in height in a population can be attributed to genetic and 20% to environmental differences. It does not mean that 80% of individual height is determined by an individual's genes. Heritability by no means implies genetic determinism (Plomin et al. 2013, pp. 93–94), as it does not say anything about the speci fic genes and causal mechanisms that produce a specific phenotypic expression (Conley et al. 2003; Johnson et al. 2009; Turkheimer 1998). Fifth, some phenotypic traits that are under strong genetic control—for instance, bipedalism—will show no heritability in standard behavioral genetic designs because there is no (or too little) variation (Shanahan et al. 2003, p. 608) as evident in (2), although they are obviously inherited.

Taken together, heritability estimates do not tell us anything about the causal mechanisms that eventually lead to an observable outcome (Turkheimer 1998). **(← p. 377)** Nonetheless, heritability has important implications for sociological explananda. If we accept that all traits are heritable to some degree, a correlation between parents and children cannot be simply seen as "prima facie evidence for sociocultural causal mechanisms" (Turkheimer 2000, p. 162). Conversely, however, heritability cannot be seen a prima facie evidence of causal genetic mechanisms.

Causal environmental influences Estimating heritability is just one possible way of exploiting the genetically sensitive twin design. One interesting implication of the idea that genetics affect all life outcomes—now general consensus in behavioral genetics (Johnson et al. 2010; Smith and Hatemi 2013; Turkheimer 2000)—is that twin designs are capable of estimating causal *environmental* influences (Johnson et al. 2010). Standard empirical research in the social sciences, which does not control for genetic endowments, implicitly assumes that

the observed correlations are not linked by genetic factors (Smith and Hatemi 2013). If social mechanisms are confounded by genetic factors, however, neglecting genetic influences will give us incorrect answers. For instance, if there is heritability in ability and schooling (as evidence shows, see below), then any assessment of how social origin impacts education and of how education impacts social outcomes will be severely biased due to unobserved genetic heterogeneity. However, by focusing on discordance in twin pairs, we open up the possibility of adjusting for (unobserved) genetic and shared environmental confounders (Johnson et al. 2009; Kohler et al. 2011). If we focus on discordance, that is, differences within twin pairs, we can estimate twin fixed effects models controlling for genetic confounding (Conley and Rauscher 2013; Fujiwara and Kawachi 2009; Kohler et al. 2011). Suppose we are interested in estimating the effect of education (x) on occupational status (y). Using information on monozygotic twins and displaying this as a regression model leads to

$$y_{ij} = \beta_{MZ} x_{ij} + A_i + D_i + I_i + C_i + E_{ij} \quad (3)$$

P is substituted by y_{ij} with the subscript i denoting family (or twin pair) and j the respective twin. As monozygotic twins are genetically identical, A_i, D_i, I_i are the same for every twin pair—as are the shared environmental influences C_i. However, this model will be biased if there is any unobserved heterogeneity in genetic or environmental influences. Focusing on discordance, a MZ twin fixed effects model as in

$$(y_{i1} - y_{i2}) = \beta_{MZ}(x_{i1} - x_{i2}) + (E_{i1} - E_{i2}) \quad (4)$$

is much less restrictive, since all genetic (A_i, D_i, I_i) and shared environmental components (C_i) drop from the equation. Thus, no assumptions on possible correlations with the independent variables are necessary, and we can estimate the effect of x on y controlling for *all* genetic and shared environmental endowments.

2.1.2 Molecular Genetics

Molecular genetic techniques examine genetic influences directly. Thus they are able to provide analysis of specific genetic influences that go beyond heritability esti-**(← p. 378)**
mates. This is supported by an increasing number of large-scale studies that have begun to provide molecular genetic data (e.g. the National Longitudinal Study of Adolescent Health (Add Health), the Panel Study of Income Dynamics, or the Framingham Heart Study; Beauchamp et al. 2011). Molecular genetic studies seek to identify specific genetically

determined biological processes affecting behavior and provide a variety of techniques to examine the relationship between genetic variation and individual differences (for an introduction, see Purcell 2013). Genetic variation between individuals is detected through genotyping. Genotyping procedures scan the entire human DNA and determine the individual's exact genotype (Purcell 2013). Two approaches that can detect these effects are being used to an increasing degree in the social sciences (see Beauchamp et al. 2011; Hatemi et al. 2011): The candidate gene association approach and the genome-wide association approach. Broadly speaking, association studies seek to pinpoint to associations between differences in individual human DNA and the trait of interest. Whereas the genome-wide association approach focuses on finding associations (quantity), the candidate gene approach is more interested in understanding the associations (quality).

As promising as it sounds to directly pinpoint the genetic variation that leads to phenotypic variations, we are far from being able to infer causal relationships. The difficulties inherent in this method result from social scientists' interest in complex traits (determined by genetic and environmental factors) rather than monogenic traits (determined by a single gene) (Guo and Adkins 2008). To date, these approaches suffer from our limited knowledge about the effects of specific candidate genes on behavioral outcomes (Conley 2009). Here, it is likely that other mechanisms are causing spurious relationships (see, i.e.; Beauchamp et al. 2011; Hatemi et al. 2011; Purcell 2013) and that results are confounded by interaction effects (between different genes or between genes and environment) that cannot be accounted for without deeper knowledge of how DNA operates. So far, results of association studies have seldom been replicated (i.e.; Beauchamp et. al 2011). As Beauchamp et al. (2011) remark in light of the difficulties entailed in measuring genotypes and phenotypes, it is important to include environmental factors. Further research has to integrate both factors, as one cannot be estimated without the other. Molecular genetics and quantitative genetics can play a complementary role in this approach, thereby producing more sensitive estimations (Kendler 2001; Weinstein et al. 2008).

2.2 Genotype–Environment Interference

The most interesting and promising pathway for integrating genetically sensitive research designs into the research on social stratification and inequality is to investigate how genes and social environment produce phenotypic outcomes in the form of gene–environment

interactions (G × E) and gene–environment correlations (rGE)[5]. **(← p. 379)**

A gene–environment interaction refers to processes by which genes alter an individual's actions towards specific features of the environment and vice versa (Shanahan and Hofer 2005). Put differently, genetic effects can vary across social groups, situations, and societies (i.e.; Johnson and Krueger 2005). The social context can operate in various ways, and so far four ideal types of G × E interactions have been differentiated (Shanahan and Hofer 2005).

The first type, triggering, means that a person has a genetic vulnerability that is expressed only in specific social situations. For example, individuals with a genetic predisposition for depression are more likely to suffer from depression when having experienced a stressful life event earlier in their lives (Silberg et al. 2001). Here the social context works detrimental and triggers the occurrence of a genetic risk.

The second type, compensation, refers to the opposite: Here, the social context is enriched and positively impacts individual functioning by hindering the expression of a genetic risk. Aggressive behavior can be prevented when growing up in intact families with warm relationships for instance (Kendler et al. 1995). Compensation and triggering do not necessarily represent an absolute dichotomy, they can rather be seen as two ends of a continuum.

In the third type, the environment serves as a mode of social control, which sounds similar to the latter but refers to (institutionalized) belief systems (i.e., norms) that are embedded in the social context. Here, individual behavior is restricted by the inherent rules of the system. The difference to compensation (i.e. avoidance of low levels of functioning) lies in the substantial mechanisms. The social control mechanism describes the limitations to individual's behavior which prevent the realization of a genetic predisposition.

The fourth type, enhancement, describes a social context that increases the genetic predisposition towards socially valued or accepted characteristics or behaviors. The difference to the first type is that enhancement refers to processes and interactions which increase positive functioning. The effect of genetic predisposition is accentuated via e.g. training or good parenting.

[5] This section describes patterns of the interplay of environmental and genetic factors. Genetic expression can be triggered by many mechanisms which are not discussed in this article. However, the newly evolving field of epigenetics provides promising insights on how environmental factors affect genes and therefore alter genetic expression without being inherited (see for a discussion on epigenetic mechanisms Shanahan and Hofer (2011)).

Other processes in which genes and environment affect each other are referred to as gene–environment correlations. Despite their name, gene–environment correlations describe a causal relation between context and behavior. A gene–environment correlation occurs when individual exposure to an environmental context depends on the genotype and vice versa (Jaffee and Price 2007). Three types of gene–environment correlations have been identified (Plomin et al. 1977): Passive, evocative, and reactive.

A passive gene environment correlation occurs when social environments appear according to inherited characteristics. Take the example of musical parents and their children. Musical parents raise their children in an environment that motivates their children to become musician themselves (i.e. instruments at home, listening to music). Being musical might also be genetically transmitted. These children passively receive a social context that fits to their genetic predisposition. An *evocative correlation* describes a situation in which genetically transmitted characteristics provoke specific reactions from the environment. For example highly talented children might receive special attention from teachers which reinforces their talents. Lastly, an **(← p. 380)** *active correlation* can be understood as a self-selection process in which individuals actively seek contexts or niches that matches their genetically-transmitted interests.

Considering both processes—gene–environment interactions as well as gene– environment correlation—will provide a more profound understanding of how the interplay of social and genetic force jointly shapes life outcomes. Gene–environment interactions reveal how genes take effect through the environment and vice versa. Gene–environment correlation comes into play when the individual genetic makeup affects environmental influences—either directly, through individual behavior, or indirectly, through selection. The existing literature clearly indicates that genes and environmental factors do not affect life outcomes independently from each other. In situations in which genetic predispositions only unfold in certain social environments, heritability estimates tend to overestimate the impact of genetic factors as they can only tell us that genes matter but not how and under which circumstances. Heritability estimations appear in this sense to be a good starting point as they indicate that social outcomes are genetically confounded. But without further investigations heritability estimation should not be over-interpreted as we do not know whether social conditions mediate these effects.

Neglecting these processes may lead to mistaken conclusions about social influences if one interprets behavior as driven solely by social causation. Acknowledging unobserved

individual genetic heterogeneity therefore substantially improves our understanding of how social inequality outcomes are shaped. Sensitive estimations have to take into account the mutual dependency between genes and environment. However, disentangling these complex patterns of genome–environment interrelationships requires interdisciplinary expertise and sophisticated research designs. Applying genetically informative designs makes it possible to go beyond a mere statistical association between genome and outcome and derive explanations based on a chain of interlinking causal factors.

3 The Relevance of Genes for Status Attainment: The Interaction of Genetic Variation and Social Mechanisms

Up to now, there have been surprisingly few genetically sensitive analyses of occupational status, one of the most frequently employed operationalizations of inequality in sociological research. The few studies that have examined the heritability of occupational status indicate that genetic factors play a substantial role in explaining individual differences in occupational status. Fulker and Eyseneck (1979) find that MZ twins are more similar in occupational status than DZ twins which indicates a heritable component. Tambs et al. (1989) replicate the heritability of occupational status across cohorts born in the first half of the twentieth century. However, both of these studies base their analyses on rather crude measurements of occupational status. Further investigations are needed to gain a precise assessment of the association between genetic factors and status attainment.

There are, however, an abundance of heritability estimates concerning psychological and physical antecedents of attainment. Most studies have focused on the heritability of cognitive skills such as IQ, with an average variation in IQ of around **(← p. 381)** 50 to 60% due to genetic influences (Bouchard and McGue 1981; Deary et al. 2009; Plomin et al. 2008). Non-cognitive abilities have been studied in the form of economic preferences (Cesarini et al. 2009; Zyphur et al. 2009) and personality traits (for a review of genetic influences on the Big Five personality traits, see Johnson et al. 2008). Considerably fewer studies focus on classical elements of the status attainment model such as educational attainment or income. A growing number of studies in this domain assesses the heritability of years of schooling (Behrman et al. 1980; Behrman and Taubman 1989; Rowe et al. 1998), examination performance in school achievement tests (Plomin et al. 2008; Bartels et al. 2002; Nielsen 2006), and broader measures of school achievement such as grades (Johnson et al. 2005, 2006, 2007). However, there is considerable variability of genetic influences on educational attainment across

different contexts (Branigan et al. 2013), indicating a complex interplay between genes and environments. A relatively large number of studies assess the genetic components of income, on average, earning correlations in the incomes of MZ twins are around 0.6 (Rowe et al. 1998; Bowles and Gintis 2002). Most recently, Benjamin et al. (2012) calculated the heritability of income: For men, 58% of 20-year income can be explained by genetic factors, compared to 46% for women.

Taken together, the current research unequivocally demonstrates that excluding the genetic component of intergenerational transmission omits an integral part of the story (Freese 2008). Nevertheless, for social inequality research to fully benefit from information on genetic variation, it is necessary to understand precisely how this information can enrich the existing theory and research. We discuss this in two steps. First, we explore the consequences of considering genetic variation either in addition to or instead of social origin in the study of status attainment. Second, we apply the formal interaction and covariance patterns presented in Sect. 2 and 3 to processes and social mechanisms discussed in the sociological status attainment research.

3.1 Social Mechanisms as Generative Processes: The Family of Origin as a Social and Genetic Point of Departure

In the research on social inequalities, parental social class, status, resources, and more recently family type are treated as the key features to assess the impact of the family of origin for later life chances. However, this convention raises theoretical as well as methodological concerns, especially for a mechanism-based explanation of status attainment. A fundamental theoretical concern is that if we want to explore how individuals maneuver themselves through the opportunity structures of a society, we need a conceptualization of individuals prior to being subjected to these socially shaped opportunities. Genetic variation offers a potential starting point. Namely, social background and other familial circumstances are already part of this opportunity structure and do not predate them (Diewald 2010). A commonly held ontological understanding of social mechanisms, as substantive mechanisms (Gross 2009; Diewald and Faist 2012), requires that a clearly defined point of departure, or cause, be distinguished from an effect and the generative processes that actively produce this effect (e.g., Machamer 2004, p. 34). Taking social origin as the starting point thus confounds cause and generative processes. This statement does not completely preclude taking social origin as a point of departure for the study of status **(← p. 382)** attainment. The argument put forward here is that

social origin is a poor concept for “origin” in a strict sense.

As we have illustrated in Sect. 2 whole genome effects as well as the effect of shared environmental influences are “black boxes” as they capture (quantify) both types of influences without having them specified. The effect of the shared environment includes family characteristics usually measured in attainment research but also those usually not measured for example infrastructural and cultural environments such as neighborhoods. Especially for young children, the shared environment estimate should closely approximate a total family effect. Thus, the systems of stratification in different societies could be described by quantifying the influence of genetic forces on the attainment process compared to shared environment or social origin (Nielsen 2006).

Heritability of attainment can be compared across subgroups (e.g. men versus women, native versus immigrants (Branigan et al. 2013), over historical time (e.g. during an economic crisis) or between national contexts (e.g. stable societies and societies in transition). Such comparisons can provide valuable information about the variability of genetic expression with respect to a specific outcome. One example is the study of the heritability of educational attainment in relation to historical changes in educational policies. According to Heath et al. (1985), parental education and genetic factors are each responsible for around 40% of the variation in educational attainment in cohorts born early in the twentieth century. Later in the twentieth century, among men, the relative importance of genetic differences increased and that of family background decreased. In women, over the same period, the heritability of educational attainment changed little. The authors attribute the increase in genetic influences to changes in educational policies that increased access to education (see also Branigan et al. (2013) for a meta-analysis of educational attainment).

Such comparisons of heritability across subgroups can be understood as relational inequality (Tilly 1998). Here, the social distribution of opportunities for attainment or for social mobility is examined by comparing the levels at which different social groups are achieving their genetic potential for success. Thus, looking at genetic variation as a cause of differential attainment fits into the broader sociological frameworks of social mechanisms that transform heterogeneities into inequalities (Diewald and Faist 2012). Higher heritability implies that genetic endowments can realize and lead to socially unhindered opportunities for attainment. Lower heritability estimates indicate that social factors limit the realization of genetic potential. A number of recent studies illustrate the variability of the genetic components of IQ depending on the socioeconomic status (SES) of the family. In low-SES families, most

variation in IQ is attributable to shared environment and very little to genetic influences. In more affluent families, this relation is reversed: Most variation in IQ is due to genetic influences and very little to the shared environment (Turkheimer et al. 2003; Nisbett et al. 2012).

Recent research has also shown how parental SES and the quality of parent–child relationships interact with genes. Social and genetic influences are interwoven from the very beginning (Chen et al. 2011; for a summary, see Shanahan 2013). In consequence, it is difficult to interpret what role social origin and other social influences play in a particular outcome such as educational attainment, because measured social **(← p. 383)** origin effects may partly reflect genetic predispositions for effort and ability as well. And ability and effort, even when measured at early ages, might not only reflect innate talent but influences of social origin. Therefore to interpret trends in attainment and mobility, several authors (Björklund et al. 2005; Jencks and Tach 2006) have emphasized the importance of studying patterns of genetic variation, arguing that if family environment is not separated from genetic relatedness, this can mask differential or even contradictory developments in gene expression in the family's social characteristics (see Branigan et al. 2013). What the "shadow of the family of origin" actually means may change over time, even if the total family effect remains the same. And if it changes, this could be due to variability in the influence of either genetic relatedness or the family's social characteristics, or both.

3.2 Patterns of Gene–Environment Interference Determining Socioeconomic Attainment

Genes matter for a person's position in society, though there is no gene for income, socioeconomic status, or social class. The only characteristics directly influenced by genes are those that lie "underneath the skin." In other words, genetically based similarities in attainment between parents and children must be explained by physical or psychological characteristics that are relevant to reach status relevant outcomes. Genetically transmitted characteristics influence individual behaviors and evoke different reactions in the environment, resulting, for example, in different labor market outcomes and recruitment to different jobs.

A common extension to better assess the effect of social origin on status attainment is to examine the impact of cognitive and non-cognitive skills, which are considered important for success in education, training, and employment (Jackson 2006; Bihagen et al. 2013; Kanfer et al. 2001). These traits, which are considered to be productivity-enhancing (Bowels et al.

2001), also have a heritable component, as described above. Other possible important characteristics which are included less often in the analyses of status attainment are physical and mental health, physical attractiveness, height, and weight. Their status as productivity-enhancing attributes is more doubtful, although they might function as such in some areas and not in others (Jackson 2006). Even less positively valued traits such as aggression may contribute to successful attainment as well. Others, such as skin color, definitely do play a role, while having no relation at all to ability or effort. This still incomplete set of very heterogeneous characteristics reveals that whole-genome effects are difficult to interpret in a substantial way.

However, the role of such personality characteristics and skills to mediate the influence of genes on socioeconomic attainment may be overestimated. As Jencks and Tach (2006, p. 38) state, "… genes are not generating intergenerational economic resemblance primarily by influencing IQ." The moderate effect of skills on the link between genes and attainment may also be due to the fact that concepts like IQ, risk aversion, time preferences, conscientiousness, and health are less proximal to genes.

An alternative strategy for studying how physical and psychological characteristics affect the interplay between genes and socioeconomic attainment is to investigate endophenotypes, which refer to more general patterns of the organism's reaction **(← p. 384)** to environmental influences that are also more proximal to genetic influences (i.e., Chen et al. 2011). Moreover, they refer to mechanisms of transcription regulation that are relevant for a broader range of developments, some of which—like behavioral problems and deviant behavior—often unobserved, despite being relevant for attainment. Shanahan (2013) provides a number of examples of a "durable programming of the stress response system", distinguishing between "fight or flight" responses to stressors. Such patterns of transcription regulation may play a crucial role in the link between social origins and socioeconomic outcomes, because on the one hand they begin to operate very early in the life course during the sensitive period around birth, with parental SES and parent–child relationships exerting a major impact on the activation or repression of genetic activity that regulates stress (Shanahan 2013). On the other hand, stress regulation appears to play a crucial role in brain development, which in turn is important for later educational and socioeconomic attainment, as reflected, for example, in a higher IQ (Nisbett et al. 2012, p. 152).

We refer to both characteristics and endophenotypes as well as to the four types of gene–environment interactions mentioned in Sect. 2: Triggering, compensation, social control, and

enhancement (Shanahan and Boardman 2009). However, we differentiate consistently between characteristics and behaviors as distinct levels at which development can occur. Characteristics and behaviors can be favorable for or detrimental to attainment. Because of this, these behavioral genetic concepts can be integrated into a more general framework of risk, risk accumulation, and risk compensation (Diewald 2011).

In life course research, risk exposure is commonly defined by the presence of risky events or episodes such as divorce, unemployment, or poverty in the life course. However, from a behavioral genetic perspective, the definition of risks starts with heterogeneity in the genetic propensity to exhibit certain "embodied" characteristics that play a role in socioeconomic attainment. These characteristics may result in either risk-averse or risk-prone behaviors or serve as criteria for institutional and organizational selection into more or less risky locations and positions. Contrary to the conventional view, this understanding of "risk" should not be confined to the emergence of negative characteristics and behaviors (e.g. aggression, anxiety). Risk also comprises a low or no propensity to exhibit favorable characteristics or the failure to realize existing genetic potential in areas such as cognitive skills or self-control. Social risks or risk compensation emerge in three steps from genetic propensities to exhibit different characteristics and behaviors:

a. as the development of favorable or detrimental physical or psychic characteristics;
b. as the manifestation of such embodied characteristics (i.e., aggression) in favorable and detrimental observed behaviors;
c. as unequal attainment resulting from these characteristics and behaviors.

In short, the blocking of detrimental characteristics and behaviors and the activation of favorable ones is good for socioeconomic attainment. Step c is then the traditional realm of sociological life course and attainment research.

To give an example of the second step: In line with the sociological adage "a gene for aggression lands you in prison if you're from the ghetto, but in the boardroom if you're to the manor born," sociologists often question whether general, geneti-**(← p. 385)**cally based traits and skills ultimately constitute important factors determining life opportunities (Conley 2009, p. 238). There are at least two possible reasons why the same genetic propensity could express itself in such divergent ways: A disposition toward aggression in upper-class children is either transformed into situation-specific, culturally accepted "know-how"—skills that make a positive difference in the sense of "power" or assertiveness—or this disposition is

effectively eliminated. The traditional thinking on the gene–environment interaction tends towards the latter interpretation, which sees this as a social control mechanism by which upper-class parents attempt to socialize their children and discourage overtly offensive behavior. But the latter interpretation may be valid as well.

The bulk of gene–environment interaction studies deal with such proximate contexts as family environment, measured as socioeconomic status (Turkheimer et al. 2003) and extensions which include family form and ethnicity (Guo and Stearns 2002). However, contexts shaping gene expression are located also at more distal levels: In neighborhoods, educational and work contexts, and societal institutions. Up to now, these multilevel interdependencies have been researched little with respect to genetic influences (for notable exceptions see Boardman et al. 2013; Branigan et al. 2013). The proximate and distal levels do not work independently of one another but may constitute chains of risk generation and risk compensation over the life course. For example, the family context may trigger or exacerbate a genetic predisposition toward deviant behavior that threatens educational success. Although this threat may be counteracted by mentoring programs in schools, such programs may fail to produce the desired long-term effects because the schools are situated in disadvantaged neighborhoods. Thus, in sum, the extent to which genetic predispositions toward specific traits that may affect socioeconomic attainment are expressed and actually affect the life course is shaped by the multilevel contexts in which individuals live, both simultaneously and successively. Nevertheless, recent interdisciplinary life course research suggests that experiences in the sensitive, very early years of life are especially important in the long run, though not in a deterministic way (Shanahan, in press). Insofar as they trigger or block genetic predispositions to traits that affect attainment repeatedly, and that are exacerbated by active as well as evocative gene–environment covariance, these experiences are decisive in cumulative advantage or disadvantage over the life course (DiPrete and Erich 2006). Genetic differences also affect the ways members of a society treat one another and how they choose their environments. Thus, there are hardly any environmental effects that are not confounded with genetic differences (gene–environment covariance; see Manuck and McCaffery 2014, p. 62).

4 Genetics and Attainment: Normative Implications

In the discussion above, we underscored that genetic information can extend our knowledge of intergenerational transmission and can help to more precisely identify social causes of attainment. In the following, we discuss how genetic information can be treated in the

framework of abstract–theoretical social mechanisms prominent in **(← p. 386)** sociological inequality research (Diewald and Faist 2012) and what normative implications this has for interpreting the genetic causes of attainment.

While the interpretation of social origin is a subject of widespread discussion in sociology, the impact of genetic variation and its interpretation in the light of equal opportunity concepts is far less discussed. Nevertheless, sometimes implicitly, sometimes explicitly, sociologists tend to interpret the whole-genome effect as "opportunity for achievement" (Nielsen 2006, p. 193), or openness of the opportunity structure: "Favorable environments, permitting fuller expression of potential, are characterized by high heritability. Unfavorable environments, inhibiting expression of native talent, are characterized by low heritability" (Nielsen 2006, p. 198). The underlying assumption is that the whole-genome effect on attainment is due to meritocratically legitimate differences in genetic endowments, and that the higher the proportion of socioeconomic attainment explained by genes, the more this genetic potential can develop without social barriers. In other words, the development and effect of talent is not restricted by social closure in access to favorable educational tracks and jobs, and not restricted by exploitation in cooperative relationships. To put it in a nutshell: In a world without social barriers, the heritability of status attainment would be 100%. Moreover, if we appreciate a society with a less restricted unfolding of genetic predispositions for socioeconomic attainment as "open", we implicitly agree that individuals must accept their good or bad luck in the gene lottery in the sense of self-ownership which means that "a person has a right to benefit from his personal genetic constitution, [because] […] it is an important part of what constitutes him as a person" (Roemer 2012, p. 484).

However, this view can be challenged in at least two respects. First, as discussed in Sect. 3, inherited traits comprise not only meritocratically legitimate talents but also skin color, height, and other ascriptive characteristics, which can by no means be seen as achievement-related and legitimate sources of inequality. If ascriptive characteristics play a decisive role, heritability does not necessarily represent openness but to some unknown extent social closure as well. From this discussion, it is evident that one should not speak of heredity as a measure of openness or "opportunity for achievement", based on meritocratically legitimate means but in a more neutral way as "opportunity for socioeconomic attainment" based on whatever inherited characteristics. Without further information, opportunities for attainment could be defined by the unrestricted realization of innate talent or by the use of stereotypes for opportunity-hoarding, or both. Only if ascriptive characteristics are removed from the whole-

genome effect by comparing the heritability of attainment in related subgroups, such as men and women, blacks and whites, migrants and non-migrants, can we approach a substantive interpretation.

Second, it can be argued against the self-ownership argument that individuals should be compensated for "bad luck" in the gene lottery since it is a fate for which they cannot be held responsible. Why should social origin, but not genetic origin, be interpreted as social closure? Is the opportunity structure more open if life chances are largely inscribed in the gene instead of being shaped by parental resources? In other words, the ultimate outcomes of the gene lottery can hardly be interpreted as pure individual achievements. From one point of view it could be argued that the gene lottery represents social closure. On the other hand individual endowments may **(← p. 387)** be able to compensate for social closure by increasing openness. To be clear: The difference between this and the former, more common interpretation is not a difference in content but in the underlying (philosophical) justice considerations (see Nussbaum 2000 for a more detailed discussion).

These normative issues are easier to discuss when using clearly defined and conjointly judged characteristics and behaviors that lead to specific socioeconomic outcomes. If genetic endowments with talent can unfold more freely in the "richer" environments of high-SES families but to a much more limited degree in low-SES families (Guo and Stearns 2002), this would indicate a need for social policy interventions. And if children's genetic propensities for stress resistance are blocked in low-SES families, producing detrimental long-term effects, a society should address this problem by compensating for unwanted social closure or by preventing it more effectively from the outset.

Finally, genetic variation alone can lead to social closure and even to social exclusion or exploitation if genetic traits are used by a society and its institutions as selection criteria. Young (1958), who coined the term meritocracy in his satirical science fiction novel "The Rise of the Meritocracy", described how genetic tests are introduced in the Great Britain of the future to screen for achievement potential. Here, contrary to any notion of openness, and justified by efficiency arguments, a favorable screening result provides subjects with an exclusive ticket to higher educational opportunities and better jobs. Yet such symbolic mechanisms are not completely unknown today. While the German school system does not make direct reference to genetic endowments, its tripartite structure is founded on the idea of providing different tracks for the different types of innate talents present in the population.

Conclusions

Can genetic variation make a significant contribution to sociological inquiry? Could the inclusion of genetic information challenge the purely social explanation of attainment and social mobility, alter the size and significance of social origin effects, or even lead to a new understanding of the social mechanisms linking social origin and destination? Our answer to these questions based on the current research is affirmative. Including genetic factors to complement and enrich the conventional way to assess social origin influences opens up new perspective in social scientific research.

The recent literature indicates that genetic and social origins play varying roles in the overall family-of-origin effect depending on historic and other contextual conditions. Nevertheless, genetically sensitive approaches stress the vital importance of different parental influences, both early as well as later in life. Sibling studies and genetically informed studies have demonstrated repeatedly that the common approach of looking at standard indicators of between-family variation in social origin captures only part of what decisively affects a child's educational and socioeconomic attainment. What is more, these measures are confounded with genetic variability, calling into question the validity and the relevance of the results (Bowles et al. 2005; Arrow et al. 2000). **(← p. 388)**

As there is still a widespread lack of rich data encompassing various social contexts as well as genetic factors, we do not yet know how much we will profit from behavioral genetics when analyzing social mobility and status attainment processes. Nevertheless, our discussion points in several potentially fruitful directions for future social mobility research. To address the complex interplay between environmental and genetic influences (Sect. 2 and 3), research should focus on gathering data that comprise a number of differently related individuals from heterogeneous social environments. This is achieved on the one hand by collecting representative data that include families and respondents from the whole range of social strata. On the other hand, the data need to be longitudinal to allow an individual to be followed across the life course through encounters with broader social contexts including neighborhoods, schools, and work environments, all of which relate to the individual's social and genetic origins and earlier experiences. The extended twin family design (ETFD), combined with molecular genetic information, offers the most promising approach to assess the interplay between social and genetic influences and how this interplay unfolds over the life course.

The benefits of disentangling the genetic and social components of the total family of origin effect are by no means restricted to social inequality research. This is not least demonstrated in demographic research and especially research on fertility behavior. Several investigations have shown that fertility is partly in our genes and that genetic and social effects depend on one another (Kohler et al 1999; Kohler and Rodgers 2003; see also the contribution of Mills and Tropf in this volume). As such this statement is not astonishing, since genetic variation may be related to genetically influenced variation in fecundity. However, genes related to fecundity can neither explain the development of fertility (and the varying contribution of genes to it) over historical time nor the changing role of educational attainment as determinant of fertility (Kohler et al. 1999). Cohort comparisons show that the difference between no parenthood and at least one child and the age at first attempt to have a child seem to be more influenced by genetic variation than the completed fertility as the number of children one gets over the life course. As Kohler et al. (1999) suggest, genetic variation contributes to fertility more over variation in preferences for parenthood than over—biological and/or material—resources to take over the responsibility for (many) children. These results are also relevant for social inequality research: If we conceive of realized fertility in the sense of unequal chances to realize preferred life goals, then we have to take into account that genetic influences on inequality may not only be due to genetic sources of resources and skills but by genetic propensities for specific preferences as well. For education and fertility there are presumably different genes at work: "overlapping sources of genetic influences are relatively small" (Kohler and Rogers 2003, p. 82). In other words, genetic variation obviously contributes to the variation in inequalities across different inequality dimensions. It can be assumed that this holds also true for different dimensions of status attainment and social mobility, namely class, status, prestige, and income.

Finally, our discussion of the empirical and normative implications of genetic variability in social stratification points to some fundamentally important issues. It is important to understand that genetic influences are far from deterministic. A high heritability estimate of an outcome does not imply that environmental factors are **(← p. 389)** unimportant. There are numerous examples that illustrate this issue: Heritability in intelligence is contingent on parental socioeconomic position (Nisbett et al. 2012), heritability of fertility depends on social context (Kohler et al. 1999; Kohler and Rodgers 2003), social control may effectively prevent genetic dispositions to aggression or drug use from unfolding (Shanahan and Hofer 2005), to cite but a few. This is vital to realize because it illustrates how environmental variability may enhance, remedy, or counter genetic influences, but it also—falsely—implies a static idea of

environment. It is misleading to think of people as genetically advantaged or disadvantaged in general. The effect of genes is always contingent on the environment—an advantage under certain conditions may be a disadvantage under others.

Moreover, the discussion of the normative issues involved in the interpretation of genetically sensitive research may enrich the long-standing discussion on the legitimacy of openness and social closure. The challenge then lies in understanding how environment and genes interact, which will bring about a refined and better understanding on how the individual is exposed to societal influences and how this affects mobility outcomes, which may ultimately contribute to the development of policies directed at increasing equality of opportunity.

References

Adkins, Daniel E., and Stephen Vaisey. 2009. Toward a unified stratification theory: Structure, genome, and status across human societies. *Sociological Theory* 27:99–121.

Arrow, Kenneth J., Samuel Bowles, and Steven N. Durlauf (Eds.). 2000. *Meritocracy and economic inequality*. Princeton University Press.

Bartels, Meike, Marjolein J. H. Rietveld, Caroline Van Baal, and Dorrit I. Boomsma. 2002. Genetic and environmental influences on the development of intelligence. *Behavior Genetics* 32:237–249.

Beauchamp, Jonathan P., David Cesarini, Magnus Johannesson, Matthijs van der Loos, Philipp D. Köllinger, Patrick J. F. Groenen, James H. Fowler, Niels J. Rosenquist, Roy A. Thurik, and Nicholas A. Christakis. 2011. Molecular genetics and economics. *Journal of Economic Perspectives* 25:57–82.

Behrman, Jere R., and Paul Taubman. 1989. Is schooling "mostly in genes"? Nature-nurture decomposition using data on relatives. *The Journal of Political Economy* 97:1425–1446.

Behrman, Jere R., Zdenek Hrubec, Paul Taubman, and Terence J. Wales. 1980. *Socioeconomic success: A study of the effects of genetic endowments, family environment, and schooling*. New York: North-Holland.

Benjamin, Daniel J., David Cesarini, Christopher F. Chabris, Edward L. Glaeser, David I. Laibson, Vilmundur Guðnason, Tamara B. Harris, Lenore J. Launer, Shaun Purcell, Albert

Vernon Smith, Magnus Johannesson, Patrik K. E. Magnusson, Jonathan P. Beauchamp, Nicholas A. Christakis, Craig S. Atwood, Benjamin Hebert, Jeremy Freese, Robert M. Hauser, Taissa S. Hauser, Alexander Grankvist, Christina M. Hultman, and Paul Lichtenstein. 2012. The promises and pitfalls of genoeconomics. *Annual Review of Economics* 4:627–662.

Bihagen, Erik, Magnus Nermo, and Charlotta Stern. 2013. Class origin and elite position of men in business firms in Sweden, 1993–2007: The importance of education, cognitive ability, and personality. *European Sociological Review* 29:939–954.

Björklund, Anders, Markus Jäntti, and Gary Solon. 2005. Influences of nature and nurture on earnings variation: A report on a study of various sibling types in Sweden. *In Unequal Chances: Family Background and Economic Success*, eds. Samuel Bowles, Herbert Gintis, Melissa Osborne Groves, 145–164. Princeton: Princeton University Press.

Blau, Peter M., and Otis D. Duncan. 1967. *The American occupational structure*. New York: Wiley and Sons.

Boardman, Jason, Jonathan Daw, and Jeremy Freese. 2013. Defining the environment in gene–environment research: Lessons from social epidemiology. *American Journal of Public Health* 103:64–72. **(← p. 390)**

Bouchard, Thomas J., and Matthew McGue. 1981. Familial studies of intelligence: A review. *Science* 212:1055–1059.

Bouchard, Thomas Jr., and Matthew McGue. 2003. Genetic and environmental influences on human psychological differences. *Journal of Neurobiology* 54:4–45.

Bowles, Samuel, and Herbert Gintis. 2002. The inheritance of inequality. *Journal of Economic Perpectives* 16:3–30.

Bowles, Samuel, Herbert Gintis, and Melissa Osborne Groves. 2001. Incentive-enhancing preferences: Personality, behavior, and earnings. *American Economic Review* 91:155–158.

Bowles, Samuel, Herbert Gintis, and Melissa Osborne Groves. 2005. *Unequal chances: family back-ground and economic success*. New York:Princeton University Press.

Branigan, Amelia R., Kenneth J. McCallum, and Jeremy Freese. 2013. Variation in the heritability of educational attainment: An international meta-analysis. *Social Forces* 92:109–140.

Breen, Richard, and Jan O. Jonsson. 2005. Inequality of opportunity in comparative perspective: Recent research on educational attainment and social mobility. *Annual Review of Sociology* 31:223–243.

Carey, James R., and James W. Vaupel. 2005. Biodemography. In *Handbook of population*, eds. Dudley Poston and Michael Micklin, 625–658. New York: Springer US.

Carmichael, Crista M., and Matt Mcgue. 1995. A cross-sectional examination of height, weight, and body mass index in adult twins. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 50:B237–B244.

Cesarini, David, Christopher T. Dawes, Magnus Johannesson, Paul Lichtenstein, and Björn Wallace. 2009. Genetic variation in preferences for giving and risk-taking. *Quarterly Journal of Economics* 124:809–842.

Chen, Edith, Gregory E. Miller, Michael S. Kobor, and Steve W. Cole. 2011. Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Molecular Psychiatry* 16:729–737.

Collins, Randall. 1979. *The credential society: An historical sociology of education and stratification*. New York: Academic Press.

Conley, Dalton. 2004. *The pecking order: A bold new look at how family and society determine who we become*. New York: Pantheon Books.

Conley, Dalton. 2009. The promise and challenges of incorporating genetic data into longitudinal social science surveys and research. *Biodemography and Social Biology* 55:238–251.

Conley, Dalton, and Emily Rauscher. 2013. Genetic interactions with prenatal social environment: Effects on academic and behavioral outcomes. *Journal of Health and Social Behavior* 54:109–127.

Conley, Dalton, Kate W. Strully, and Neil G. Bennett. 2003. *The starting gate. Birth weight and life chances*. Berkeley: University of California Press.

Coventry, William L., and Matthew C. Keller. 2005. Estimating the extent of parameter bias in the classical twin design: A comparison of parameter estimates from extended twin family and classical twin designs. *Twin Research and Human Genetics* 8:214–223.

Deary, Ian. J., Wendy Johnson, and Michael Houlihan. 2009. Genetic foundations of human intelligence. *Human Genetics* 126:215–232.

Derks, Eske M., Conor V. Dolan, and Dorret I. Boomsma. 2006. A test of the equal environment assumption (EEA) in multivariate twin studies. *Twin Research and Human Genetics* 9:403–411.

Diewald, Martin. 2010. Zur Bedeutung genetischer Variation für die soziologische Ungleichheitsforschung. *Zeitschrift für Soziologie* 39:4–21.

Diewald, Martin. 2011. Neither conflicting nor simply complementary: How social inequality research can profit from paying attention to genetic variation. In *Equal is not enough: challenging differences and inequalities in contemporary societies. Conference Proceedings*, eds. Joz Motmans, Daniel Cuypers, Petra Meier, Dimitri Mortelmans, Patrizia Zanoni, 334–351. Antwerp: Policy Research Centre on Equal opportunities: University of Antwerp-Hasselt University.

Diewald, Martin, and Thomas Faist. 2012. *From heterogeneities to inequalities: Looking at social mechanisms as an explanatory approach to the generation of social inequalities. SFB 882 Working Paper Series, No. 1. Bielefeld*: DFG Research Center.

DiPrete, Thomas, and Gregory M. Eirich. 2006. Cumulative advantage as a mechanism for inequality: A review of theory and evidence. *Annual Review of Sociology* 32:271–297.

Downey, Douglas B. 1995. When bigger is not better: Family size, parental resources, and children's educational performance. *American Sociological Review* 60:746–759.

Erikson, Robert, and John H. Goldthorpe. 1992. The constant flux: *A study of class mobility in industrial societies*. Oxford: Oxford University Press. **(← p. 391)**

Freese, Jeremy. 2008. Genetics and the social science explanation of individual outcomes. *American Journal of Sociology* 114:1–35.

Fujiwara, Takeo, and Ichiro Kawachi. 2009. Is education causally related to better health? A twin fixed-effect study in the USA. *International Journal of Epidemiology* 38:1310-1322.

Fulker, David W., and Hans J. Eyseneck. 1979. Nature and nurture: Heredity. In *The structure and measurement of intelligence*, eds. Hans J. Eyseneck. New York: Springer.

Gross, Neil. 2009. A pragmatist theory of social mechanisms. *American Sociological Review* 74:358–379.

Guo, Guang, and Elisabeth Stearns. 2002. The social influences on the realization of genetic potential for intellectual development. *Social Forces* 80:881–910.

Guo, Guang, Yuying Y. Tong, and Tianji J. Cai. 2008. Gene by social context interactions for number of sexual partners among white male youths: Genetics-informed sociology. *American Journal of Sociology* 114:36–66.

Haller, Archibald O., and Alejandro Portes. 1973. Status attainment processes. *Sociology of Education* 46:51–91.

Hatemi, Peter K., Christopher T. Dawes, Amanda Frost-Keller, Jaime E. Settle, and Brad Verhulst. 2011. Integrating social science and genetics: News from the political front. *Biodemography and Social Biology* 57:67–87.

Hauser, Robert M., and Raymond S. Wong. 1989. Sibling resemblance and intersibling effects in educational attainment. *Sociology of Education* 62:149–171.

Hauser, Robert M., John R. Warren, Min-Hsiung Huang, and Wendy Y. Carter. 2000. Occupational status, education, and social mobility in the meritocracy. In *Meritocracy and economic inequality*, eds. Kenneth Arrow, Samual Bowles, and Steven Durlauf, 179–229. Princeton: Princeton University Press.

Heath, Andrew C., Alexandre A. Todorov, Elliot C. Nelson, Pamela A. F. Madden, Kathleen K. Bucholz, and Nicholas G. Martin. 2002. Gene-environment interaction effects on behavioral variation and risk of complex disorders: The example of alcoholism and other psychiatric disorders. *Twin Research* 5:30–37.

Heckman, James J. 2006. Skill formation and economics of investing in disadvantaged children. *Science* 312:1900–1902.

Jackson, Michelle. 2006. Personality traits and occupational attainment. *European Sociological Review* 22:187–199.

Jaffee, Sara R., and Thomas S. Price. 2007. Gene-environment correlations. A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry* 12:432–442.

Jencks, Christopher, and Laura Tach. 2006. Would equal opportunity mean more mobility? In *Mobility and inequality: Frontiers of research in sociology and economics*, eds. Stephen Morgan, David Grusky, and Gary Fields, 23–58. Stanford: Stanford University Press.

Johnson, Wendy, and Robert F. Krueger. 2005. Genetic effects on physical health: Lower at higher income levels. *Behavior Genetics* 35:579-590.

Johnson, Wendy, Kirsten O. Kyvik, Erik L. Mortensen, Axel Skytthe, G. David Batty, and Ian J. Deary. 2010. Education reduces the effects of genetic susceptibilities to poor physical health. *International journal of epidemiology* 39:406-414

Johnson, Wendy, Matt McGue, and William G. Iacono. 2005. Disruptive behavior and school achievement: Genetic and environmental relationships in 11-year-olds. *Journal of Educational Psychology* 97:391–405.

Johnson, Wendy, Matt McGue, and William G. Iacono. 2006. Genetic and environmental influences on academic achievement trajectories during adolescence. *Developmental Psychology* 42:514–532.

Johnson, Wendy, Matt McGue, and William G. Iacono. 2007. Socioeconomic status and school grades: Placing their association in broader context in a sample of biological and adoptive families. *Intelligence* 35:526–541.

Johnson, Andrew M., Philip A. Vernon, and Amanda R. Feiler. 2008. Behavioral genetic studies of personality: An introduction and review of the results of 50+ years of research. In *The Sage handbook of personality theory and assessment*, eds. Gregory J. Boyle, Gerald Matthews, and Donald H. Saklofske, 145–173. Thousand Oaks: Sage Publications.

Johnson, Wendy, Eric Turkheimer, Irving I. Gottesman, and Thomas J. Bouchard. 2009. Beyond heritability: Twin studies in behavioral research. *Current Directions in Psychological Science* 18:217–220.

Kanfer, Ruth, Connie R. Wanberg, and Tracy M. Kantrowitz. 2001. Job search and employment: A personality-motivational analysis and meta-analytic review. *Journal of Applied Psychology* 86:837–855.

Keller, Matthew C., Sarah E. Medland, Laramie E. Duncan, Peter K. Hatemi, Michael C. Neale, Hermine H. M. Maes, and Lindon J. Eaves. 2009. Modeling extended twin family data

I: Description of the cascade model. *Twin Research and Human Genetics* 12:8–18. **(← p. 392)**

Kendler, Kenneth S. 2001. Twin studies of psychiatric illness: An update. *Archives of General Psychiatry* 58:1005–1014.

Kendler, Kenneth S., Ronald C. Kessler, Ellen E. Walters, Charles MacLean, Michael C. Neale, Andrew C. Heath, and Lindon J. Eaves. 1995. Stressful life events, genetic liability, and onset of an episode of major depression in women. *American Journal of Psychiatry* 152:833–42.

Kim, Yong-Kyu. 2009. *Handbook of behavior genetics*. New York: Springer.

Kohler, Hans-Peter, and Joseph L. Rodgers. 2003. Education, fertility, and heritability: Explaining a paradox. In Offspring: *Human fertility behavior in biodemographic perspective*, ed. National Research Council of the National Academies, 46–90. The National Academies Press. Washington D.C.

Kohler, Hans-Peter., Joseph L. Rodgers, and Kaare Christensen. 1999. Is fertility behavior in our genes? Findings from a Danish twin study. *Population and Development Review* 25:253–288.

Kohler, Hans-Peter, Jere R. Behrman, and Jason Schnittker. 2011. Social science methods for twins data: Integrating causality, endowments, and heritability. *Biodemography and Social Biology* 57:88–141.

Machamer, Peter. 2004. Activities and causation: The metaphysics and epistemology of mechanisms. *International Studies in the Philosophy of Science* 18:27–39.

Manuck, Stephen B., and Jeanne M. McCaffery. 2014. Gene-environment interaction. Annual *Review of Psychology* 65:41–70.

Nielsen, François. 2006. Achievement and ascription in educational attainment: Genetic and environmental influences on adolescent schooling. *Social Forces* 85:193–216.

Nisbett, Richard E., Joshua Aronson, Clancy Blair, William Dickens, James Flynn, Diane F. Halpern, and Eric Turkheimer. 2012. Intelligence. New findings and theoretical developments. *American Psychologist* 67:130–159.

Nussbaum, Martha. 2000. Review of from chance to choice: Genetics and justice by Allen

Buchanan, Dan W. Brock, Norman Daniels and Daniel Wikler. *New Republic* 12:38–48.

Plomin, Robert, and Denise Daniels. 2011. Why are children in the same family so different from one another? *International Journal of Epidemiology* 40:563-582.

Plomin, Robert, John C. DeFries, and John C. Loehlin.1977. Genotype-environment interaction and correlation in the analysis of human development. *Psychological Bulletin* 84:309–322.

Plomin, Robert, John C. DeFries, Gerald E. McClearn, and Peter McGuffin. 2008. *Behavioral genetics*. 5th. New York: Worth Publishers.

Plomin, Robert, John C. DeFries, Valerie S. Knopik, and Jenae M. Neiderhiser. 2013. *Behavioral genetics*. 6th. New York: Worth Publisher

Plomin, Robert, and Stephen M. Kosslyn. 2001. Genes, brain and cognition. *Nature neuroscience* 4:1153-1154.

Posthuma, Daniëlle. 2009. Multivariate genetic analysis. In *Handbook of behavior genetics*, eds. Yong-Kyu Kim, 47–60. New York: Springer.

Posthuma, Dorret, and Danielle I. Boomsma. 2000. A note on the statistical power in extended twin designs. *Behavior Genetics* 30:147–158.

Purcell, Shaun. 2013. Appendix: Statistical methods in behaviorial genetics. In *Behavioral genetics*, eds. Robert Plomin, John C. DeFries, Valerie S. Knopik, and Jenae M. Neiderhiser 6th. New York: Worth Publishers.

Roemer, John. 2012. What is the justification of studying intergenerational mobility of economic status? In *From parents to children*, eds. John Ermisch, Markus Jäntti, and Timothy Smeeding, 482–488. New York: Russell Sage Foundation.

Rowe, David C., Wendy J. Vesterdal, and Joseph L. Rodgers. 1998. Herrnstein's syllogism: Genetic and shared environmental influences on IQ, education, and income. *Intelligence* 26:405–423.

Rutter, Michael, Michael Pickles, Andrew Murray, and Lindon Eaves. 2001. Testing hypotheses on specific environmental causal effects on behavior. *Psychological Bulletin* 127:291–324.

Saunders, Peter. 2002. Reflections on the meritocracy debate in Britain: A response to Richard Breen and John Goldthorpe. *British Journal of Sociology* 53:559–574.

Scarr, Sandra, and Louise Carter-Saltzman. 1979. Twin method: Defense of a critical assumption. *Behavior Genetics* 9:527–542.

Shanahan, Michael J. 2013. Social genomics and the life course: Opportunities and challenges for multilevel population research. In *New directions in the sociology of aging*, eds. Linda J. Waite and Thomas J. Plewes, 255–276. Washington D.C.: The National Academies Press.

Shanahan, Michael J., and Jason Boardman. 2009. Genetics and behavior in the life course: a promising frontier. In *The craft of life course research*, eds. Glen A. Jr. Elder and Janet Z. Giele, 215–235. London: Guilford. **(← p. 393)**

Shanahan, Michael J., and Scott M. Hofer. 2005. Social context in gene-environment interactions: Retrospect and prospect. *Journals of Gerontology* 60B:65–76.

Shanahan, Michael. J., and Scott M. Hofer. 2011. Molecular genetics, aging, and well-being: sensitive period, accumulation, and pathway models. In *Handbook of aging and social sciences*, eds. Robert

H. Binstock and George L. K. Morris, 135–147. New York: Elsevier.

Shanahan, Michael J., Scott M. Hofer, and Lilly Shanahan. 2003. Biological models of behavior and the life course. In *Handbook of the Life Course - Handbooks of Sociology and Social Research*, eds. Jeylan T. Mortimer and Michael J. Shanahan, 597-622. US: Springer.

Silberg, Judy L., Michael Rutter, and Lindon Eaves. 2001. Genetic and environmental influences on the temporal association between earlier anxiety and later depression in girls. *Biological psychiatry* 49:1040-1049.

Smeeding, Timothy, Robert Erikson, and Markus Jäntti. 2011. Introduction. In *Persistence, privilege, and parenting*, eds. Timothy Smeeding, Robert Erikson, and Markus Jäntti, 1–26. New York: Russell Sage Foundation.

Smith, Kevin B, and Peter K. Hatemi. 2013. OLS is AOK for ACE: a regression-based approach to synthesizing political science and behavioral genetics models. *Political Behavior* 35:383-408.

Tambs, Kristian, Jon Martin Sundet, Per Magnus, and Kare Berg. 1989. Genetic and environmental contributions to the covariance between occupational status, educational attainment, and IQ: A study of twins. *Behavior Genetics* 19:209–222.

Tilly, Charles. 1998. *Durable inequality*. Berkeley: University of California Press.

Turkheimer, Eric. 1998. Heritability and biological explanation. Psychological Review 105:782-791.

Turkheimer, Eric. 2000. Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science* 9:160-164.

Turkheimer, Eric, Andreana Haley, Mary Waldron, Brian D'Onofrio, and Irving I. Gottesman. 2003. Socioeconomic status modifies heritability of IQ in young children. *Psychological Science* 14:623–628.

Vaupel, James W. 2004. The biodemography of aging. *Population and Development Review* 30:48–62.

Visscher, Peter M., William G. Hill, and Naomi R. Wray. 2008. Heritability in the genomics era—concepts and misconceptions. *Nature Reviews Genetics* 9:255–266

Weinstein, Maxime, James Vaupel, and Kenneth Wachter. 2008. Biosocial surveys. *Report of the National Research Council of the National Academies*. Washington D.C.: The National Academies Press.

Zyphur, Michael J., Jayanth Narayanan, Richard D. Arvey, and Gordon J. Alexander. 2009. The genetics of economic risk preferences. *Journal of Behavioral Decision Making* 22:367–377.

Martin Diewald, 1958, Prof. Dr. Phil., Fakultät für Soziologie an der Universität Bielefeld, Sprecher des SFB 882 "Von Heterogenitäten zu Ungleichheiten" und Ko-Leiter des DFG-Langfristvorhabens "TwinLife – Genetic and social causes of life chances". Forschungsgebiete: Soziale Ungleichheiten, Lebenslaufforschung, Verhaltensgenetik. Veröffentlichungen: Zur Bedeutung genetischer Variation für die soziologische Ungleichheitsforschung. Zeitschrift für Soziologie 39, 2010; The Sociology of the Life Course

and Life Span Psychology. Integrated Paradigm or Complementing Pathways. In: Advances in Life Course research. Special Issue: Reconsidering the Linked Life Principle. Amsterdam 2009 (mit K.U. Mayer).

Tina Baier, 1984, M.A., wissenschaftliche Mitarbeiterin an der Universität Bielefeld/ DIW-Berlin im DFG-Langfristvorhaben "TwinLife". Forschungsgebiete: soziale Ungleichheit, Bildungsungleichheit und Methoden kausaler Inferenz. Veröffentlichungen: Die Auswirkung von Studiengebühren auf die Studierneigung in Deutschland: Evidenz aus einem natürlichen Experiment auf Basis der HIS-Studienberechtigtenbefragung (mit M. Helbig und A. Kroth). Zeitschrift für Soziologie 41, 2012.

Wiebke Schulz, 1983, Dr., wissenschaftliche Mitarbeiterin an der Universität Bielefeld/ DIW-Berlin im DFG-Langfristvorhaben "TwinLife – Genetic and social causes of life chances". Forschungsgebiete: soziale Ungleichheiten, historische Soziologie, Lebenslaufforschung. Veröffentlichungen: Career Chances and Constraints in the Netherlands 1865-1940 (mit I. Maas). European Sociological Review 28, 2012; Employer's choice – Selection through job advertisements in the nineteenth and twentieth centuries. Research in Social Stratification and Mobility, 2014 (mit I. Maas & M.H.D. van Leeuwen). **(← p. 394)**

Reinhard Schunck, 1979, Jun-Prof. Dr., Fakultät für Soziologie an der Universität Bielefeld. Forschungsgebiete: Lebenslaufforschung, soziale Ungleichheiten, Gesundheit, Migration und Integration, quantitative Methoden. Veröffentlichungen: Pathways between Perceived Discrimination and Health among Immigrants. Ethnicity and Health. DOI 10.1080/13557858.2014.932756, 2014 (mit K. Reiss und O. Razum); Within- and Between-Estimates in Random Effects Models. Advantages and Drawbacks of Correlated Random Effects and Hybrid Models. The Stata Journal 13, 2013. **(← p. 395)**